

Leptin (LEP) Antibody (C-term)
Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP6150a

Specification

Leptin (LEP) Antibody (C-term) - Product Information

Application	WB, IF, E
Primary Accession	P41159
Other Accession	NP_000221
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Antigen Region	134-166

Leptin (LEP) Antibody (C-term) - Additional Information

Gene ID 3952

Other Names

Leptin, Obese protein, Obesity factor, LEP, OB, OBS

Target/Specificity

This Leptin (LEP) antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 134-166 amino acids from the C-terminal region of human Leptin (LEP).

Dilution

WB~~1:1000

IF~~1:10~50

E~~Use at an assay dependent concentration.

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.

Storage

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

Leptin (LEP) Antibody (C-term) is for research use only and not for use in diagnostic or therapeutic procedures.

Leptin (LEP) Antibody (C-term) - Protein Information

Name LEP ([HGNC:6553](#))

Function Key player in the regulation of energy balance and body weight control. Once released

into the circulation, has central and peripheral effects by binding LEPR, found in many tissues, which results in the activation of several major signaling pathways (PubMed:[15899045](#), PubMed:[17344214](#), PubMed:[19688109](#)). In the hypothalamus, acts as an appetite-regulating factor that induces a decrease in food intake and an increase in energy consumption by inducing anorexinogenic factors and suppressing orexigenic neuropeptides, also regulates bone mass and secretion of hypothalamo- pituitary-adrenal hormones. In the periphery, increases basal metabolism, influences reproductive function, regulates pancreatic beta-cell function and insulin secretion, is pro-angiogenic for endothelial cell and affects innate and adaptive immunity (By similarity) (PubMed:[11460888](#), PubMed:[19688109](#), PubMed:[24340098](#), PubMed:[25060689](#), PubMed:[8589726](#)). In the arcuate nucleus of the hypothalamus, activates by depolarization POMC neurons inducing FOS and SOCS3 expression to release anorexinogenic peptides and inhibits by hyperpolarization NPY neurons inducing SOCS3 with a consequent reduction on release of orexigenic peptides (By similarity). In addition to its known satiety inducing effect, has a modulatory role in nutrient absorption. In the intestine, reduces glucose absorption by enterocytes by activating PKC and leading to a sequential activation of p38, PI3K and ERK signaling pathways which exerts an inhibitory effect on glucose absorption (PubMed:[24340098](#)). Acts as a growth factor on certain tissues, through the activation of different signaling pathways increases expression of genes involved in cell cycle regulation such as CCND1, via JAK2-STAT3 pathway, or VEGFA, via MAPK1/3 and PI3K-AKT1 pathways (By similarity) (PubMed:[17344214](#)). May also play an apoptotic role via JAK2-STAT3 pathway and up-regulation of BIRC5 expression (PubMed:[18242580](#)). Pro-angiogenic, has mitogenic activity on vascular endothelial cells and plays a role in matrix remodeling by regulating the expression of matrix metalloproteinases (MMPs) and tissue inhibitors of metalloproteinases (TIMPs) (PubMed:[11460888](#)). In innate immunity, modulates the activity and function of neutrophils by increasing chemotaxis and the secretion of oxygen radicals. Increases phagocytosis by macrophages and enhances secretion of pro-inflammatory mediators. Increases cytotoxic ability of NK cells (PubMed:[12504075](#)). Plays a pro-inflammatory role, in synergy with IL1B, by inducing NOS2 which promotes the production of IL6, IL8 and Prostaglandin E2, through a signaling pathway that involves JAK2, PI3K, MAP2K1/MEK1 and MAPK14/p38 (PubMed:[15899045](#), PubMed:[19688109](#)). In adaptive immunity, promotes the switch of memory T-cells towards T helper-1 cell immune responses (By similarity). Increases CD4(+)CD25(-) T-cell proliferation and reduces autophagy during TCR (T-cell receptor) stimulation, through MTOR signaling pathway activation and BCL2 up-regulation (PubMed:[25060689](#)).

Cellular Location

Secreted.

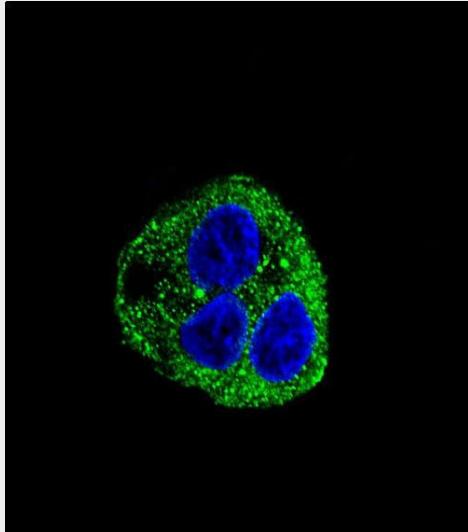
Tissue Location

Adipose tissue is the main source of leptin. It is also produced by other peripheral tissues such as the skeletal muscle (PubMed:12448771, PubMed:16052473, PubMed:7789654). Expressed by intercalated and striated tracts of submandibular and parotid salivary gland intralobular ducts (PubMed:12448771). Detected by fundic epithelium of the gastric mucosa (PubMed:10896907). Secreted into blood and gastric juice (PubMed:10896907).

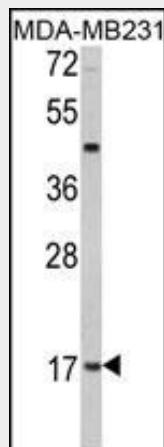
Leptin (LEP) Antibody (C-term) - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

Leptin (LEP) Antibody (C-term) - Images

Confocal immunofluorescent analysis of Leptin (LEP) Antibody (C-term)(Cat#AP6150a) with HepG2 cell followed by Alexa Fluor® 488-conjugated goat anti-rabbit IgG (green). DAPI was used to stain the cell nuclear (blue).



Western blot analysis of hLep-R149 (Cat. #AP6150a) in MDA-MB231 cell line lysates (35ug/lane). LEP (arrow) was detected using the purified Pab.

Leptin (LEP) Antibody (C-term) - Background

Leptin (LEP) is an adipocyte-secreted protein that regulates body fat and endocrine functions. Leptin is similar to the mouse obesity protein encoded by the *obese* gene (*ob*). In mouse studies, mutations in this gene are linked to severe and morbid obesity.

Leptin (LEP) Antibody (C-term) - References

- Tank, J., et al., *J. Clin. Endocrinol. Metab.* 88(10):4955-4959 (2003).
- Silha, J.V., et al., *Eur. J. Endocrinol.* 149(4):331-335 (2003).
- Kazumi, T., et al., *Metab. Clin. Exp.* 52(9):1113-1116 (2003).
- Mullington, J.M., et al., *J. Neuroendocrinol.* 15(9):851-854 (2003).
- Catalano, S., et al., *J. Biol. Chem.* 278(31):28668-28676 (2003).